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Competing risks analysis of lamb mortality in a terminal sire composite population[1]

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ABSTRACT: Mortality records from birth to weaning of 8,301 lambs from a composite population at the U.S. Meat Animal Research Center were analyzed using a competing risks model. The advantage of the competing risks model over traditional survival analyses is that different hazards of mortality can be assigned to different causes, such as disease, dystocia, and starvation. In this study, specific causes of mortality were grouped into dam-related (DAMR; e.g., dystocia and starvation), pneumonia (PNEU), disease (DIS; excluding pneumonia), and other (OTHER) categories. The hazard of mortality was analyzed using a competing risk approach, where each mortality category was assumed to be independent. Continuous- and discrete-time survival analyses were implemented using sire, animal, and maternal effects mixed models. The continuous-time survival analysis used the Weibull model to describe the hazard of mortality for each category of mortality. Under the discrete-time survival analysis, a complementary log-log link function was used to analyze animal-time data sets using weekly intervals for each category of mortality. Explanatory variables were sex, type of birth, contemporary group, and age of dam. The significant influences of type of birth and age of dam effects were consistent across category of mortality, and the sex effect was significant for all categories except the OTHER category. Estimates of variance components indicated strong maternal effects for all categories except for PNEU. Estimates of additive genetic heritabilities from the discrete maternal effects models were 0.08 ± 0.04, 0.09 ± 0.18, 0.16 ± 0.12, 0.19 ± 0.09, and 0.14 ± 0.10 for OVERALL (all causes combined), DIS, DAMR, PNEU, and OTHER categories, respectively. Ignoring the cause of the defining event in mortality and longevity studies may hide important genetic differences. Therefore, the effectiveness of breeding programs relying on models that ignore multiple causes of an event in time-to-event data, such as mortality and longevity, could be affected.

Key Words: Heritability, Population Mortality, Sheep, Survival Analysis

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Introduction

Lamb mortality is an example of time-to-event data characterized by the length of time until mortality. Lamb mortality can be due to a variety of reasons, including disease, exposure, dystocia, starvation, injury, and poisoning. The cause of mortality is often ignored when estimating genetic parameters (e.g., Mukasa-Mugerwa et al., 2000; Southey et al., 2001; Nguti et al., 2003). This approach assumes that there is a single hazard of mortality, although the lambs may have different hazards of mortality associated with different causes of mortality (Allison, 1997).

Numerous studies have concluded that there is substantial genetic variation for the various causes of mortality. Mortality differences in sheep breeds associated with different causes have been reported (Nash et al., 1997; Mukasa-Mugerwa et al., 2000; Nguti et al., 2003). Genetic variation in cause of mortality has been reported by Lush et al. (1948) and Robertson and Lerner (1949) in poultry, and by Gama et al. (1991) in sheep. Dürr et al. (1999 and 2002) and Roxström and Strandberg (2002) identified genetic variability in dairy cow survival associated with different removal reasons. Therefore, the cause of mortality must be included when modeling mortality.

Competing risks analysis is an approach to account for cause of mortality when modeling mortality. A competing risk analysis considers that the competition among the different causes of mortality results in the

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Table 1. Summary statistics for number of records, mean age at mortality, standard deviation of age at mortality (SD), and mortality rate by each mortality category

		Mortality		
Category	No.	Age, d	SD	Rate, %
Overall	1,217	11.3	13.7	14.7
Dam-related	308	3.9	6.9	3.7
Disease	176	12.3	12.3	2.1
Pneumonia	359	21.8	13.9	4.3
Other	374	6.8	12.2	4.5

observed mortality by one of the different mortality causes. Thus, a lamb that is alive has an associated risk of mortality due to each mortality cause. With the occurrence of mortality due to one cause, the risks of mortality due to the other causes are removed. The objective of this study was to assess the environmental and genetic factors associated with different causes of lamb mortality using complementary models in a competing risks approach.

Materials and Methods

Description of Population and Management

Mortality records were obtained on lambs from F_3 and advanced generations of a terminal sire composite population (breed composition: 50% Columbia, 25% Hampshire, and 25% Suffolk) at the U.S. Meat Animal Research Center, Clay Center, Nebraska. Leymaster (1991) and Mousa et al. (1999) have previously described the breed formation and management of this population. Lamb records were available from 1985 through 1997 and included the date of mortality and cause of mortality determined by necropsy. General descriptions of the different sheep mortality causes can be found in the Sheep Industry Handbook (American Sheep Industry Association, 2003). Records from lambs that lost their identification were removed because the status of these lambs was unknown. Records from lambs raised in the nursery were removed because the nursery environment was expected to provide a different risk than the dam environment. The data consisted of 8,301 lambs from 299 sires and 2,475 dams.

Causes of mortality from birth to weaning were grouped into the following categories: disease (**DIS**; excluding pneumonia), dam-related (**DAMR**; including dystocia and starvation), pneumonia (**PNEU**), and other (**OTHER**; including eating disorders, injury, weather related, physical reasons, and unspecified causes). These categories were consistent with biological considerations of individual causes and had a minimum number of records necessary to obtain precise estimates. The overall (**OVERALL**) category included mortality by any cause and corresponded with the mortality definition used by Southey et al. (2001, 2003). A summary of the number of records by cause of mortality is given in Table 1.

Statistical Analyses

Lamb mortality or failure time accounting for cause of mortality was analyzed using a competing risks approach by assigning a hypothetical potential or latent failure time to each category of mortality. Each category of mortality has an unobserved or latent time of failure that can occur provided that the other category of mortality cannot occur. The observed mortality time is the minimum of these latent failure times and results in the observed category of mortality. After the occurrence of mortality by one category, the nonminimum times associated with the other categories are no longer observable. Thus, the latent failure times associated with categories other than the observed category of mortality are considered to be right-censored at observed mortality because the mortality associated with these categories must be larger than the observed mortality (Crowder, 2001). A hypothetical illustration of this concept considers that the latent failure times for a lamb from DAMR, DIS, PNEU, and OTHER categories are 3, 15, 40, and 50 d, respectively. The observed failure time or mortality record for this lamb would be 3 d, because this is the minimum of the latent failure times. This observed mortality record would be treated as observed for the DAMR category, but as censored at a value of 3 d for the DIS, PNEU, and OTHER categories.

Given the available information on time, cause of the event, and time-independent explanatory variables, it is not possible to distinguish between an independent competing risk model and any cause-specific hazard model (Crowder, 2001; Kalbfleisch and Prentice, 2002). To overcome the identifiability problem inherent to competing risks models, the causes of mortality were assumed to act independently; thus, the latent failure times are treated as independent (Crowder, 2001; Kalbfleisch and Prentice, 2002). This assumption was made for this analysis because there is no additional information on the potential differences between causes to justify any specific dependency.

The likelihood function of continuous-time mortality records can be factored into independent cause-specific likelihood functions associated with each competing risk (Allison, 1997; Crowder, 2001; Kalbfleisch and Prentice, 2002). Within cause, the mortality records from other causes are treated as censored observations at the time of the mortality (Allison, 1997; Crowder, 2001; Kalbfleisch and Prentice, 2002). Therefore, the competing risks model for continuous-time mortality can be implemented using standard survival analysis.

The likelihood function of discrete-time mortality records cannot be factored in a manner similar to the continuous-time mortality model, and requires the simultaneous estimation of all cause specific models (Allison, 1997; Crowder, 2001). Allison (1997) described a procedure to analyze each cause separately based on the multinomial analysis approach of Begg and Gray (1984). This approach was implemented using a modification of the discrete-time approach described by

Southey et al. (2003) by creating separate animal-time data sets for each cause of mortality. In each data set, the mortality record of every lamb was initially divided into discrete 1-wk intervals. The number of discrete time intervals varied between causes to avoid a potential extreme value problem (Misztal et al., 1989), with very few or no mortality recorded in an interval. The number of discrete-time intervals was 4 wk for the DAMR category, 6 wk for DIS category, and 7 wk for the PNEU and OTHER categories. Each interval consisted of a binary response variable that indicated either the occurrence or nonoccurrence for the specific cause of mortality. For example, a lamb that died in the third week due to PNEU would have three time intervals in the PNEU category data set, where the first two time intervals would be recorded as nonoccurrence of mortality and the third time interval would be recorded as occurrence of mortality due to PNEU. The same lamb in a data set for another category would consist of the first two time intervals being recorded as nonoccurrences of mortality and the third time interval recorded as censored.

A Weibull model was used for the continuous-time mortality records using the generalized linear mixed model class in Matvec (Kachman and Fernando, 2002). A complementary log-log link function was used for the analysis of discrete-time mortality records in ASREML (Gilmour et al., 1999) because the coefficients have the same relative risk interpretation as coefficients from the Cox proportional hazards model (Allison, 1997; Kalbfleisch and Prentice, 2002). The explanatory variables for both approaches were sex, age of dam (four levels: 1, 2, 3, and 4+ yr of age), type of birth (three levels: single-, twin-, and triplet or higher-born), and contemporary group (18 levels derived from year of lambing and breeding period within year). In addition, the discrete time interval was included as an explanatory variable in the discrete-time approach. Sire, animal, and maternal effects models were fitted for both approaches. The maternal effects model includes additive genetic variances of direct effects, additive genetic effects for maternal effects, and the possible covariance between the direct additive genetic and maternal effects. Heritabilities were computed as $\sigma^2_g/(\sigma^2_g + \pi^2/6)$, where σ^2_g is the additive genetic variance and $\pi^2/6$ is the variance of the Weibull model and complementary log-log function (Fahrmeir and Tutz, 1994). The formula to compute the standard error of the heritability is provided in the ASREML manual (Gilmour et al., 1999).

Results

Estimates of the explanatory variable effects were virtually identical between the sire, animal, and maternal discrete-time models; hence, only estimates of the hazard ratios from the animal model are presented (Table 2). The hazard ratios in the OVERALL category of mortality, ignoring individual mortality categories, are interpreted as follows: Male lambs had 24% ($P < 0.001$) greater hazard of mortality than female lambs. Single- and twin-born lambs had 75 and 60% ($P < 0.001$) lower hazard, respectively, than triplet or higher-born lambs. Lambs from 1-yr-old and 2-yr-old dams had 200 and 39% ($P < 0.001$) greater hazard than lambs from dams 4-yr of age and older, whereas lambs from 3-yr-old dams had 18% ($P < 0.001$) lower hazard than lambs from older dams.

The difference in hazard ratios among mortality categories provides support for the rationale of considering cause in the analysis of lamb mortality. The estimated hazard ratio varied among mortality categories although only lamb sex showed a marked difference from the trend observed in all categories (Table 2). Male lambs had a hazard of mortality similar to female lambs in the OTHER category; however, male lambs had significantly ($P < 0.001$) greater hazard of mortality than female lambs in the PNEU and DAMR categories. Male lambs had a nonsignificantly greater hazard than female lambs for mortality in the DIS category.

Triplet- or higher-born lambs had significantly ($P < 0.001$) greater hazard of mortality than single- or twin-born lambs, and twin-born lambs had significantly ($P < 0.001$) greater hazard of mortality than single-born lambs in all categories. The highest hazard of mortality associated with type of birth occurred in the DAMR category, with single- and twin-born lambs having 81 and 71% lower hazard of mortality than triplet or higher-born lambs, respectively. The lowest hazard of mortality associated with type of birth occurred in the DIS and PNEU categories, with single-born lambs having 68 and 59% lower hazard of mortality than triplet- or higher-born lambs respectively, and twin-born lambs having 40 and 44% lower hazard of mortality than triplet or higher-born lambs, respectively.

Lambs from 3-yr-old dams had a risk of mortality similar to lambs from dams 4 yr of age and older in all categories. The OVERALL hazard of mortality of lambs from 2-yr-old dams was greater than of lambs from dams 4 yr and older but was not significantly different in any individual category. Lambs from 2-yr-old dams had significantly higher risk of mortality ($P < 0.01$) than lambs from 3-yr-old dams except in the DIS category, with the differences being marginal ($P < 0.10$). One-year-old dams provided a significantly ($P < 0.001$) poorer maternal environment because their lambs had a greater hazard of mortality than lambs from older dams.

The probability of lamb mortality varied depending on the discrete time interval and was greatest in the first week of life for all categories (Figure 1). The probability of lamb mortality, after adjusting for the terms in the model, during the first week was 9.8% in the OVERALL category, indicating that for the average lamb, there was a 10% probability of mortality in the first week. The probability of mortality in the OVERALL category was under 2% from the second week of age until weaning.

Table 2. Hazard ratios and standard errors for the explanatory variables by cause of mortality

	Mortality category				
Effect[a]	Overall	Disease	Dam-related	Pneumonia	Other
Lamb sex					
M – F	1.24 ± 0.07	1.28 ± 0.20	1.40 ± 0.16	1.48 ± 0.16	0.92 ± 0.10
Type of birth					
1 – 3+	0.25 ± 0.03	0.32 ± 0.10	0.19 ± 0.04	0.41 ± 0.09	0.24 ± 0.05
2 – 3+	0.40 ± 0.04	0.60 ± 0.15	0.29 ± 0.05	0.56 ± 0.11	0.40 ± 0.06
Age of dam					
1 – 4+	3.00 ± 0.40	3.02 ± 1.11	3.68 ± 1.07	2.40 ± 0.57	2.83 ± 0.66
2 – 4+	1.39 ± 0.17	1.53 ± 0.51	1.63 ± 0.43	1.08 ± 0.23	1.47 ± 0.31
3 – 4+	0.82 ± 0.10	1.08 ± 0.36	1.03 ± 0.28	0.74 ± 0.16	0.67 ± 0.15

[a]M – F = effect of male lambs as deviation from female lambs; 1 – 3+ = effect of single-born lambs as a deviation from triplet or higher-born lambs; 2 – 3+ = effect of twin-born lambs as a deviation from triplet or higher-born lambs; 1 – 4+ = effect of 1-yr-old ewes as a deviation of 4-yr-old or older ewes; 2 – 4+ = effect of 2-yr-old ewes as a deviation of 4-yr-old or older ewes; 3 – 4+ = effect of 3-yr-old ewes as a deviation of 4-yr-old or older ewes.

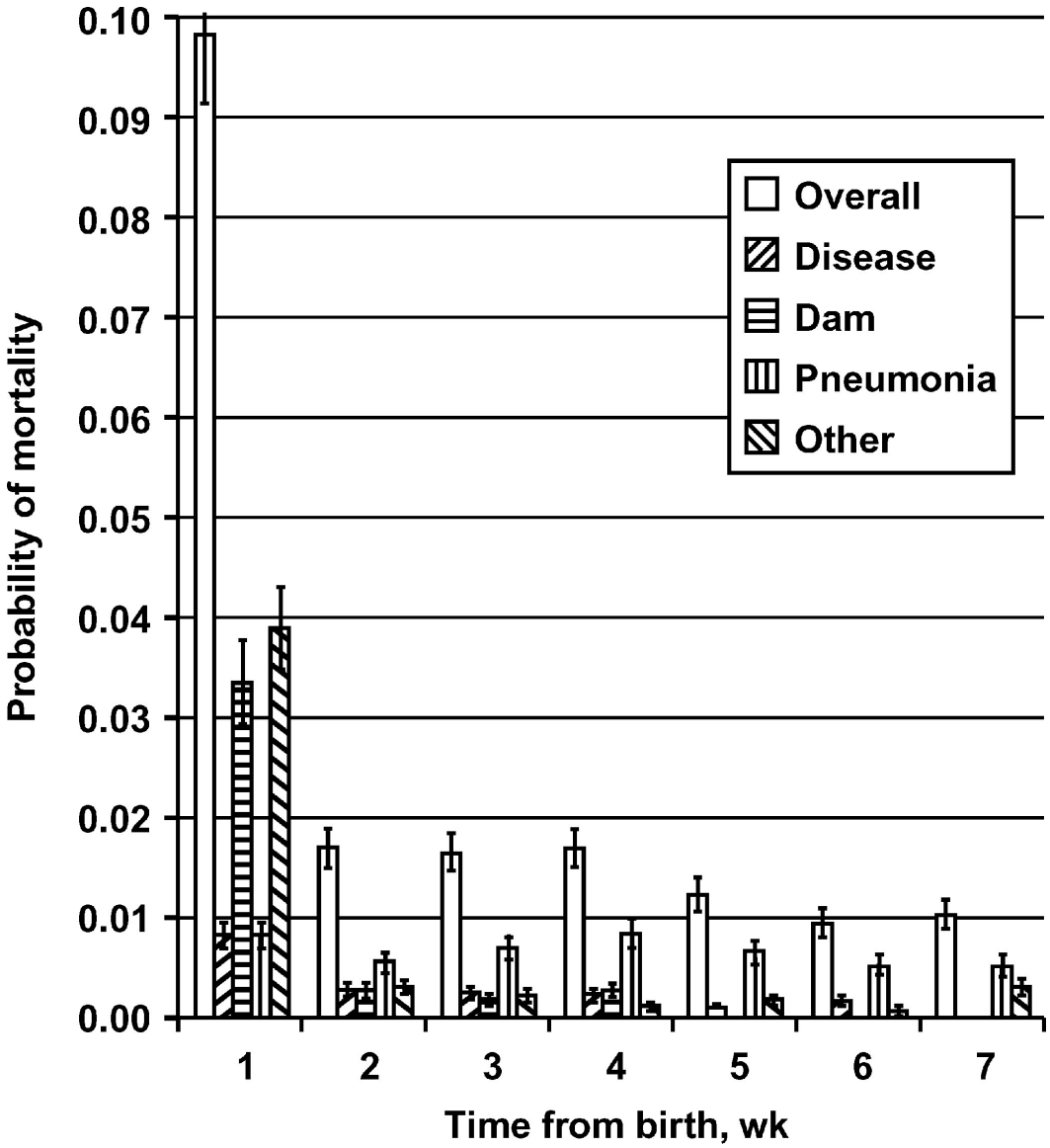

Figure 1. Probability of mortality and standard errors from birth to weaning for each cause of mortality.

Table 3. Estimates of variance components and associated heritabilities from sire and animal continuous-time models by mortality category

	Mortality category				
Model	Overall	Disease	Dam-related	Pneumonia	Other
Sire model					
Sire variance	0.068 ± 0.022	0.130 ± 0.128	0.371 ± 0.109	0.135 ± 0.072	0.149 ± 0.074
Heritability	0.159 ± 0.050	0.293 ± 0.267	0.736 ± 0.177	0.304 ± 0.150	0.332 ± 0.151
Animal model					
Additive genetic variance	0.802 ± 0.112	0.316 ± 0.330	1.146 ± 0.292	0.328 ± 0.350	0.998 ± 0.323
Heritability	0.328 ± 0.031	0.161 ± 0.141	0.411 ± 0.062	0.166 ± 0.148	0.378 ± 0.076

The adjusted probabilities of mortality showed different patterns among the individual categories from birth to weaning (Figure 1). Of the individual categories, mortality in the DAMR category was highest in the first week (3.2%), but it dropped below 0.3% in the second week and was virtually nonexistent after the fourth week of age. In contrast, mortality in the PNEU category was almost constant for all 7 wk, ranging between 0.5 and 0.8%. Overall mortality dropped from 9.8% in the first week to 1.7% in the second wk and gradually decreased to 1% by weaning. Consequently, OVERALL mortality did not entirely reflect the changes in the different categories during the period studied.

The variance and associated heritability estimates for different categories are presented in Table 3 for the continuous-time approach and in Table 4 for the discrete-time approach. Similar estimates of sire variance and heritability were obtained for the OVERALL cause and individual categories for the continuous- and discrete-time approaches using the sire model. In general, the continuous-time approach using the animal model provided greater estimates of additive genetic variances and, hence, greater heritability estimates than the discrete-time approach. The maternal effects model only converged for the continuous-time analysis of OVERALL and PNEU causes, assuming a zero correlation between direct and maternal effects. For the OVERALL category, the estimated direct additive variance was 0.450 ± 0.119 and the estimated maternal variance was 0.206 ± 0.073, resulting in an additive heritability estimate of 0.213 ± 0.048. For the PNEU category, the estimated direct additive variance was 0.539 ± 0.228 and the estimated maternal variance was 0.112 ± 0.153, resulting in an additive heritability estimate of 0.235 ± 0.080.

The heritability estimates varied with the model and category of mortality considered. The high estimates of the additive genetic heritability observed in the sire and animal models for mortality in the DAMR category suggest large maternal effects. This interpretation is supported by the smaller direct additive genetic variance in maternal effects models compared with estimates from the sire and animal models (Table 4). The estimates of direct additive heritability in the animal and maternal effects models indicated that there is genetic variation within these categories. There was substantial maternal genetic variation across mortality categories, with the largest variation found in the DIS category. The estimates of correlation between direct and maternal effects varied with mortality category and had large standard errors. There was insufficient information in the data to estimate this parameter precisely. Assuming zero covariance between direct additive genetic and maternal effects resulted in slightly higher

Table 4. Estimates of variance and covariance components and associated heritabilities and correlations from sire, animal, and maternal discrete-time models by mortality category

	Mortality category				
Model	Overall	Disease	Dam-related	Pneumonia	Other
Sire model					
Sire variance	0.054 ± 0.021	0.130 ± 0.127	0.337 ± 0.105	0.131 ± 0.071	0.119 ± 0.070
Heritability	0.128 ± 0.047	0.293 ± 0.266	0.679 ± 0.175	0.295 ± 0.149	0.270 ± 0.148
Animal model					
Additive genetic variance	0.232 ± 0.073	0.221 ± 0.305	0.603 ± 0.216	0.538 ± 0.195	0.421 ± 0.173
Heritability	0.124 ± 0.034	0.118 ± 0.144	0.268 ± 0.070	0.246 ± 0.067	0.204 ± 0.067
Maternal effects model					
Additive genetic variance (A)	0.154 ± 0.081	0.183 ± 0.384	0.353 ± 0.266	0.424 ± 0.228	0.292 ± 0.203
Maternal variance (M)	0.198 ± 0.085	0.649 ± 0.435	0.376 ± 0.260	0.027 ± 0.186	0.526 ± 0.224
Covariance (A, M)	–0.057 ± 0.077	–0.182 ± 0.375	–0.074 ± 0.250	0.102 ± 0.175	–0.206 ± 0.201
Additive genetic heritability	0.082 ± 0.043	0.087 ± 0.181	0.159 ± 0.119	0.185 ± 0.094	0.142 ± 0.100
Maternal heritability	0.105 ± 0.046	0.307 ± 0.225	0.169 ± 0.125	0.012 ± 0.081	0.257 ± 0.117
Correlation (A, M)	–0.325 ± 0.355	–0.527 ± 0.791	–0.204 ± 0.600	0.941 ± 4.419	–0.526 ± 0.350

heritability estimates. The largest changes occurred with the PNEU category, where the direct additive heritability changed from 0.185 ± 0.094 to 0.210 ± 0.083, and the maternal heritability changed from 0.012 ± 0.081 to 0.046 ± 0.066. The estimates from the discrete-time maternal effect model with a zero covariance between direct additive genetic and maternal effects were similar to the corresponding estimates from the maternal effect model fitted in the continuous-time approach.

Discussion

The estimates of the explanatory variable effects on OVERALL lamb mortality in this study were similar to previously reported estimates (Southey et al., 2001, 2003). The influence of birth type and age of dam on mortality were generally consistent across the categories. Thus, the effect of these factors on mortality was not dependent on the cause of mortality. In contrast, the effect of sex varied with the cause of mortality and this result is consistent with the literature. Sex differences have been reported in traits associated with the maternal category, including length of parturition (Alexander et al., 1993; Cloete et al., 2002) and time from standing to sucking (Alexander et al., 1993; Cloete and Scholtz, 1998). However, no sex differences were observed in cold resistance (Slee et al., 1991).

The influence of the maternal environment in this study was in agreement with Gama et al. (1991), who analyzed different definitions of lamb mortality, including a respiratory cause, from birth to weaning. Gama et al. (1991) reported low heritability estimates (<10%) of mortality using a half-sib model, but higher estimates of heritability for total mortality from birth to weaning (30%) and birth to 24 h (89%) under full-sib model. Gama et al. (1991) reported similar estimates of heritability associated with respiratory causes between half-sib and full-sib approaches. This suggested that, in agreement with this study, there was minimal maternal influence on respiratory causes of mortality.

Gama et al. (1991) used a cumulative binary approach, where the response variable was defined as occurrence or nonoccurrence. In contrast, this study used a discrete-time approach that Southey et al. (2003) had shown to provide similar estimates of variance components to the continuous-time and cumulative binary analysis reported by Southey et al. (2001). This cumulative binary approach could lead to biased parameter estimates in survival analysis with a single cause of mortality due to different degrees of censoring within the levels of an explanatory variable. Ingram and Kleinman (1989) showed that the cumulative binary analysis provided large biases when censoring only occurred in one level of a two level factor but showed no bias when censoring occurred in both levels of the factor. Consequently, the cumulative binary analysis applied to the data sets used in this study requires additional caution compared with the discrete-time analysis due to the increased censoring that occurs by treating mortality from other causes as censored.

The estimates of additive genetic variance suggest that identifying different categories of mortality would permit potential selection against specific causes of interest. Estimates of genetic parameters and selection experiments indicate that selection on mortality due to the factors that contribute to the DAMR category can be effective in decreasing mortality in this category. Cloete et al. (2002) reported low heritability estimates for lambing and neonatal behavior characteristics, and Hinch (1997) found substantial breed differences. Selection for improved rearing ability has also improved lamb survival (Atkins, 1980; Donnelly, 1982; Haughey, 1983). Cloete and Scholtz (1998) observed that lambs from the high rearing ability line had higher survival rates and were quicker to progress from standing to sucking than lambs from the low rearing ability line. Indirect response to selection has also increased pelvic size (Knight et al., 1988; Kilgour and Haughey, 1993), thus reducing lambing difficulty. Bennett (2001) reported decreased calving difficulty in cattle selected for improved calving ease.

Cold resistance as an indicator of exposure is another important determinant of lamb survival in many environments. Although this aspect was not directly considered in this study, there have been reports of genetic variation. Experiments with cold resistance using water bath tests have reported heritability estimates of 0.3 (Slee and Stott, 1986), 0.36 (Wolff et al., 1987), and 0.7 (Slee et al., 1991). Slee and Stott (1986) reported a realized heritability of 0.27 for increased cold resistance but a realized heritability of zero for decreased cold resistance. In addition, a major gene associated with cold resistance has been identified (Slee and Simpson, 1991; Simpson and Slee, 1998).

The competing risks approach used here assumed that the categories of mortality are independent, even though some of the categories may not be strictly independent, such as PNEU and OTHER diseases. The grouping of individual causes into broader categories can result in loss of information when compared with the study of specific individual causes. However, the grouping is expected to increase the power if the grouped causes share the genetic mechanism of control. For example, the Nramp locus in mice is known to provide resistance to a variety of diverse and unrelated pathogens (Vidal et al., 1993).

The magnitude of the correlations between breeding value estimates from different mortality categories was used as an indicator of the suitability of the independence of categories assumption. The correlations between the individual categories were close to zero (range = –0.12 to 0.19) and smaller than the correlation between each individual category and the OVERALL category (range 0.18 to 0.50). Thus, the response to selection for mortality, regardless of cause, is likely to be limited because few animals would be favorable for several causes. The low magnitudes of the correlations

also suggest that the assumption of independent causes may be appropriate since high correlations would be expected if the causes were indicators of the same response variable. However, the use of breeding values to assess independence is challenging due to bias associated with censoring (Vukasinovic et al., 1999), the lack of identifiability in competing risks, and because only the minimum mortality time of the potential failure times is recorded.

The estimates of the variance components for OVERALL mortality are in agreement with our previous reports (Southey et al., 2001, 2003) applying continuous- and discrete-time survival analysis. These similarities between the different approaches were expected from the results of Ingram and Kleinman (1989) and Doksum and Gasko (1990) and suggest that a variety of approaches using different software can be applied to study mortality. Our studies have shown that there is genetic variation for lamb survival assuming either a single cause or multiple causes. The estimates of individual categories support the suggestion of Cundiff et al. (1982) that selection of components of mortality is likely to be more effective than selection solely on mortality, regardless of cause.

Implications

This study has shown the value of applying a competing risks model to survival data. The multiple factors involved in mortality indicate that ignoring the cause of the event may hide important genetic differences. The results from this study suggest that there are opportunities to change management and selection practices to improve lamb mortality that are specific to the mortality cause. This conclusion is supported by experimental evidence from selection line studies that show that lamb survival was changed by selection for improved rearing ability, a maternal aspect of lamb mortality. Therefore, breeding programs are likely to be ineffective when multiple causes involved in time-to-event data, including mortality and longevity, are ignored. Consequently, recording and analysis of different causes of mortality are justified if breeding goals involve the improvement of lamb survival.

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